**Amendments to the Specification:**

1. Please replace the paragraph beginning on page 11, line 30, with the following amended paragraph:

The inventors have found that BiP suppressed aggregation *in vivo*. In particular, a mutant BiP that is unable to release substrate (T19G BiP), caused a larger pool of SMA to be retained within the ER and decreased the frequency of aggresome formation. The inventors also found that co-expression of Hsp70 with SMA decreases the frequency of aggresome formation. Without ALLN (SEQ. ID. NO. 11) treatment 30% of the COS cells contained aggresomes as assayed by immunofluorescence. Addition of ALLN (SEQ. ID. NO. 11) increased the proportion to 75%, but co-expression of Hsp70 dramatically decreased the frequency of aggresomes to 12% whether or not ALLN (SEQ. ID. NO. 11) was present.

2. Please replace the paragraph beginning on page 12, line 7, with the following amended paragraph:

To further investigate the effects of Hsp70 and BiP on the aggregation of SMA *in vivo*, the inventors used a biochemical assay. The inventors found that forced interaction with these chaperones improves the fate of SMA. When BiP or T19G BiP were co-expressed with SMA, a modest effect on the pool size of soluble LC was observed, as compared to cells transfected with SMA alone. This effect was variable in the absence of ALLN (SEQ. ID. NO. 11), but consistently observed in the presence of ALLN. (SEQ. ID. NO. 11) Co-expression of either of these chaperones had no effect on the size of the insoluble SMA pool.

3. Please replace the paragraph beginning on page 13, line 2, with the following amended paragraph:

The inventors also found that An Hsp70-binding peptide derived from the LC sequence inhibits SMA aggregation in vivo. To optimize delivery of the peptide to all cellular compartments, it was synthesized with the 11-mer sequence from the HIV TAT protein at the N-terminus (Gius et al., 1999). This TAT peptide (SEQ. ID. NO. 8) permits the transduction of denatured proteins across cell membranes rapidly and efficiently in an energy- and receptor-independent fashion. In addition to the test peptide, TAT-TISS (SEQ. ID. NO. 9), another TAT-fusion was employed as a specificity control. This peptide, TAT-PASS (SEQ. ID. NO. 10), contains four amino acid substitutions and does not inhibit fibril formation *in vitro*. SMA transfected cells were incubated overnight in the presence of increasing concentrations of ALLN (SEQ. ID. NO. 11) and 50 μM of each peptide. In the range of 1-10 μg/ml ALLN, (SEQ. ID. NO. 11) there was a progressive increase in the amount of SMA found in the soluble fraction on a per cell basis. Inclusion of the TAT-TISS (SEQ. ID. NO. 9) peptide dramatically reduced the amount of SMA recovered at all ALLN (SEQ. ID. NO. 11) concentrations tested. In contrast, the TAT-PASS (SEQ. ID. NO. 9) peptide had no effect. Incubation of the same blots with anti-raf antibody demonstrated that equal cell equivalents were loaded across the gel.

4. Please replace the paragraph beginning on page 13, line 15, with the following amended paragraph:

The inventors also determined the effect of different concentrations of peptide on SMA following treatment with 10 μg/ml ALLN. (SEQ. ID. NO. 11) The TAT-TISS (SEQ. ID. NO. 9) peptide decreased the yield of SMA in the insoluble fractions much more than in the detergent soluble fractions. The magnitude of the decrease was from 4 to 10-fold (n=3), in a peptide concentration-dependent fashion, whereas the TAT-PASS (SEQ. ID. NO. 10) peptide had only a marginal effect even at the highest concentration used. As observed with co-expression of Hsp70, upon addition of the TAT-TISS (SEQ.

ID. NO. 9) peptide, the ubiquitinated forms of SMA were diminished. This indicated that they were being maintained in a soluble form long enough to be kept off the aggregation pathway and were degraded by the proteasome.

5. Please replace the paragraph beginning on page 13, line 23, with the following amended paragraph:

Lastly, the inventors determined that the decrease in steady state level of SMA in the presence of TAT-TISS (SEQ. ID. NO. 9) peptide correlated with a decrease in the frequency of aggresome formation, by scoring anti-kappa stained cells. Roughly 30% of untreated cells exhibited aggresomes and this number increased to about 65% upon addition of ALLN. (SEQ. ID. NO. 11) Transduction of TAT-TISS (SEQ. ID. NO. 9) decreased the number of aggresomes by more than half, to 25%, about the same as in untreated cells, whereas addition of TAT-PASS (SEQ. ID. NO. 10) peptide had no significant effect. Hence, the large decrease in insoluble SMA observed in the presence of TAT-TISS (SEQ. ID. NO. 9) peptide coincides with a drop in aggresome formation.